MUCOSAL HEALING AFTER HIGH DOSE PANTOPRAZOLE IN ACUTE CORROSIVE INJURY OF ESOPHAGUS

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CERTIFICATE

This is to certify that the dissertation entitled **"MUCOSAL HEALING AFTER HIGH DOSE PANTOPRAZOLE IN ACUTE CORROSIVE INJURY OF ESOPHAGUS"** is a bonafide work done by **Dr.N.A.Rajesh**, Madras Medical College in partial fulfilment of the university rules and regulations for the award of DM Medical Gastroenterology under my guidance and supervision during the academic year August 2011-2014.

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DECLARATION

I solemnly declare that this dissertation entitled **"MUCOSAL HEALING AFTER HIGH DOSE PANTOPRAZOLE IN ACUTE CORROSIVE INJURY OF ESOPHAGUS"** was done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, during 2011-2014 under the guidance and supervision of **Prof.MOHAMMED ALI M.D., D.M.** This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfilment of requirements for the award of D.M. Degree in Medical Gastroenterology (Branch-IV).

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CONTENTS

INTRODUCTION

The ingestion of corrosive agents is still an important public health issue in our country.

The GIT injuries caused by caustic agents can range from minor to fatal, or can lead to chronic disease with poor quality of life.

Corrosive agents with a pH level less than two or more than twelve can rapidly penetrate the layers of the oesophagus and result in necrosis and scar formation in the mucosa.[1]

Acidic agents produce coagulation necrosis and eschar formation that may limit tissue penetration and may even spare the oesophagus when the transit is rapid.

On the other hand, Alkaline agents when ingested produce liquefaction necrosis and can cause serious oesophageal injury by penetrating to deep muscle layers.[2]

The basic histopathologic reaction of tissue subjected to caustic burn is the synthesis, deposition and remodelling of collagen. Following full-thickness injuries to the oesophageal wall, the normal oesophagus is replaced by dense connective tissue. Collagen overproduction has been estimated to cause stenosis in half of the patients suffering severe burns.[3]

Consequently, when treating caustic burn injuries, it is necessary to prevent stenosis by inhibiting collagen synthesis or changing the properties of the deposited collagen.

The optimal management protocol in the treatment of severe caustic injury remains controversial.

The main goal of medical treatment is to inhibit inflammatory reaction or stricture formation secondary to oesophageal burning. Stricture formation is thought to be overcome by suppressing fibroplasia and scarring.

Many agents directed at wound healing and stricture prevention have been used in several experimental studies in past.[4-7] Results of such treatment protocols including steroids, antibiotics, heparin, indomethacin, sucralfate, vitamin E, as well as total parenteral nutrition are all controversial in treatment of corrosive burns.[4–9]

Randomised control trials on the role of proton pump inhibitors in caustic injuries of GIT are lacking. A few experimental studies have investigated the relationship between proton pump inhibitors and corrosive burns and has shown that proton pump inhibitors can reduce inflammation in early phase of caustic injury.[8]

Proton pump inhibitors, by decreasing gastric acid secretion and thereby GER can prevent worsening of corrosive injury. In addition proton pump inhibitors has also been shown to have anti-inflammatory and antioxidant properties.[10,11]

A prospective study from turkey on 13 patients have showed that omeprazole can be used effectively in treatment of acute corrosive injury of esophagus.[12] There are no studies from India which have attempted to assess the usefulness of proton pump inhibitors in treatment of acute corrosive injury of esophagus. This prompted us to study the usefulness of pantoprazole in the treatment of patients presenting with acute corrosive injury of the oesophagus.

AIM OF THE STUDY

To study the efficacy of high dose pantoprazole in causing mucosal healing after acute corrosive injury of esophagus.

REVIEW OF LITERATURE

Acute corrosive poisonings result from ingestion of acids, bases, heavy metal salts, oxidants, and other chemical substances.

Chemical substances with strong caustic features are commonly used in industries, households and everyday life.

The production and widespread usage of caustic agents with high concentration and different (high) pH value have increased the incidence of corrosive poisoning.

TABLE 1.1: COMMONLY INGESTED CAUSTIC SUBSTANCES

Caustic substance	Type	Commercially available form
Acids	Sulfuric	Batteries Industrial cleaning agents Metal plating
	Oxalic	Paint thinners, strippers Metal cleaners
	Hydrochloric	Solvents Metal cleaners Toilet and drain cleaners Antirust compounds
	Phosphoric	Toilet cleaners
Alkali	Sodium hydroxide	Drain cleaners Home soap manufacturing
	Potassium hydroxide	Oven cleaners Washing powders
	Sodium carbonate	Soap manufacturing Fruit drying on farms
Ammonia	Commercial ammonia	Household cleaners
	Ammonium hydroxide	Household cleaners
Detergents, bleach	Sodium hypochlorite	Household bleach, cleaners
	Sodium polyphosphate	Industrial detergents
Condy’s crystals	Potassium permanganate	Disinfectants, hair dyes

EPIDEMIOLOGY

Corrosive injuries are largely under reported in India and other developing countries.

National poisoning statistics, are lacking in India and hence authentic data on corrosive poisoning is lacking.

In United states the incidence of caustic injury is more than 26,000 cases per year.

Caustic injuries are common in children, and are mostly accidental.

Since these agents have become an usual household item, young children usually under 5 years of age, due to their inquisitive and exploring nature accidentally ingest these substances resulting in corrosive injuries. This is particularly true in developing countries where overcrowding and unhygienic living conditions combined with poor regulatory control expose children to such chemicals.

The most reported corrosive agents are caustic soda, sodium hypochlorite and other alkaline household chemicals.[13]

Acid ingestion is more common in India than in other countries.[14]

A multicentric study of children from India showed that 1.7%–9.3% of all cases of poisoning were due to corrosive ingestion, ranking behind kerosene, drugs and pesticide poisonings.[15]

Some authors have shown that mucosal injury to the esophagus is more serious and severe injuries are more frequently observed in patients who attempted suicide as compared with accidental ingestion.[16]

The estimated national suicide rate for India in 2005 was 10.3/100000 population.

Tamil Nadu state has a higher rate than the national average with 18.6 suicides per 100 000 population, while Puducherry has the highest rate of suicides at 52.1/100 000 population.[17]

Data on suicides are available from Tamilnadu as verbal autopsy reports of large population cohorts.[18] These data group corrosive ingestion along with other poisons and do not provide a split up of the data to determine the frequency of suicidal deaths attributable to corrosive ingestion.

Therefore, we have to rely on data of patients admitted with corrosive poisoning to tertiary care centres to determine the proportion of corrosive poisonings which are suicidal.

Table 1.2: EPIDEMIOLOGICAL DATA ON CORROSIVE POISOINIG FROM INDIA

Author,place (year)	No.(n)	Mean age(yrs)	Males (%)	Acid ingestion(%)	Suicidal intent (%)
Rao et al[19], Puducherry (1988)	50	2%Children	46.0	68.0	54.2
Zargar et al[20] Chandigarg (1989)	41	26.0	66.7	100	39.0
Poddar,Thapa[21] Chandigarg (2001)	54	4.9	77.7	62.9	0
Gupta,[22] Chandigarg (2004)	51	26.5	66.6	83.4	na
Ananthakrshnan[23] Puducherry (2010)	109	4-65	55.0	82.6	na

na- not available

Suicidal corrosive injuries are often associated with marked oral, oropharyngeal and proximal oesophageal burns because of hesitant sipping of the fluid whereas accidental injuries are usually associated with ingestion of larger volumes which are gulped down fast and are associated with a higher proportion of gastric injuries.

The principle that 'Acid licks the oesophagus and bites the stomach' has been challenged by many authors. It has been shown that even with acid ingestion oesophageal injuries are common.[24]

PATHOPHYSIOLOGY

Acids and alkalis produce different types of tissue damage. Coagulative necrosis, with eschar formation is the hallmark of acid induced tissue damage. Eschar formation acts as a limiting factor in tissue penetration and depth of injury[25].

On the other hand, alkalis by combining with tissue proteins cause liquefactive necrosis and saponification. Higher viscosity causing longer contact time, facilitate deeper penetration into oesophageal tissues. In addition, absorption of alkali produce thrombosis of submucosal blood vessels thereby impeding blood flow to already damaged tissue[26].

The extent of tissue destruction depends on the premorbid state of the tissue, pH of the agent ,quantity, physical form and concentration of the caustic agent.

Alkali ingestion may lead to more serious esophageal injury and complications, however this distinction is probably not relevant in the setting of strong acid ingestion. Strong acids also can penetrate tissues rapidly, leading to full-thickness damage of the esophageal wall.

Extensive esophageal damage and perforations after acid ingestion has been reported[27].

Liquid agents are more injurious than the granular corrosives because the granules often adhere to the mucous membranes of the mouth preventing further movement into the esophagus.

Caustic Injury occurs quickly after ingestion, depending on the agent's concentration and duration of exposure[28], a 30% solution of sodium hydroxide can produce full thickness injury in 1 s[29].

Previous studies have shown that the requisite pH for esophageal injury is 12.5 (0.4% sodium hydroxide has a pH of 13).[30]

The severest location of corrosive injury of esophagus generally occurs in the narrowest portion , at the level of the aortic arch.

Initially, tissue injury is marked by eosinophilic necrosis with swelling and hemorrhagic congestion[31]. Experimental findings suggest that arteriolar and venular thrombosis with consequent ischemia may be more important in the pathogenesis of acute corrosive injury[32].

Mucosal sloughing and bacterial invasion occur four to seven days after ingestion .At 1 week granulation tissue appears, and ulcers become covered by fibrin. Perforation may occur during this period if ulceration exceeds the muscle plane.

Esophageal repair usually begins on the tenth day , whereas ulcerations begin to reepithelialise approximately 1 month after exposure. Collagen deposition begins after 2nd week and the tensile strength of the healing tissue is low during the first three weeks. Therefore, endoscopy and dilatation is preferably avoided 5-15 d after ingestion[33].

Scar retraction begins by the third week and continues for several months, resulting in stricture formation and shortening.

The lower esophageal sphincter pressure becomes impaired, increases gastroesophageal reflux and accelerates stricture formation[34].

Pathologically, within 10 days of caustic esophageal injury, granulation tissue begins to replace the necrotic epithelium, and by 21 days fibroblasts start producing epithelial strictures.

CLINICAL PRESENTATION

Clinical presentation depends on the physical state, type and quantity of the corrosive substance.

Corrosive agents in powder or crystal state adhere to oral cavity and throat, causing severe oropharyngeal injury as opposed to the liquid agents that pass rapidly through the oesophagus.

70% of patients with oropharyngeal injury do not have significant oesophageal involvement.[35]Therefore oropharangeal burns are not reliable index of esophageal injury.

Similarly ,absence of oropharyngeal injury does not exclude severe injuries of the other areas of the gastrointestinal tract.

Hypersalivation, dysphagia, edema, ulceration or whitish plaques in the oral cavity, palatal mucosa and pharynx are common phenomena[36, 37, 38].

(i) Acute Phase

Injuries of the larynx may cause laryngospasm associated with dyspnoea, tachypnea, aphonia and dysphonia.

Hoarseness of voice indicates laryngeal, epiglottic or hypopharyngeal complications.

Aspiration of the corrosive substance can cause endotracheal and bronchial necrosis with mediastinitis, leading to death[39].

High temperature accompanied with fever suggests perforation.

Painful and burning mouth and throat, retrosternal chest and stomach pains, hemetemesis are frequently present.

These symptoms may occur immediately or may be delayed for few hours after ingestion and last for days to weeks.

Severe corrosive injuries of the stomach may result in perforation and development of acute abdomen. This requires emergency surgery.

These injuries may appear in the first 2 days or may be delayed until the 14thday after corrosive ingestion[40].

(ii) Late complications

Late complications are a major problem corrosive poisoning and often cause permanent handicap in patients.

The most common late complications are oesophageal strictures and stenosis, gastric antral and pyloric stenosis , & oesophageal and stomach cancer[41].

Strictures and stenosis of the esophagus – may appear three weeks after ingestion of the corrosive substance, in the first three months or even after one year.

Stenosis of antrum and pylorus – patients presenting with feeling of fullness of stomach, nausea, vomiting, and weight loss suggests the presence of gastric outlet obstruction.

Esophageal and stomach cancer – the latent period from the ingestion of the corrosive substance and the development of cancer may range between 40 to 50 years.

EVALUATION AND ASSESSMENT

(i) Laboratory studies

A high WBC count (> 20000 cells/mm3), elevated serum CRP, older age and the presence of an esophageal ulcer have been considered predictors of mortality in adults[42]

An arterial pH < 7.22 or a base excess < -12 is considered indication of severe esophageal injury and of emergency surgery[43].

However, laboratory studies are more useful in monitoring and guiding treatment rather than predicting morbidity or mortality[44].

(ii) Traditional radiology

Plain chest radiograph useful to rule out perforation, may suggest aspiration pneumonitis

(iii) Endoscopic Ultrasound

EUS using a miniprobe to evaluate esophageal wall seems safe, however does not show any difference with endoscopy in predicting early complications[45]. The destruction of the muscular layers of the oesophagus is a reliable sign of future stricture formation[46].

EUS may predict the response to dilatation. If the muscolaris propria is involved more sessions are required.

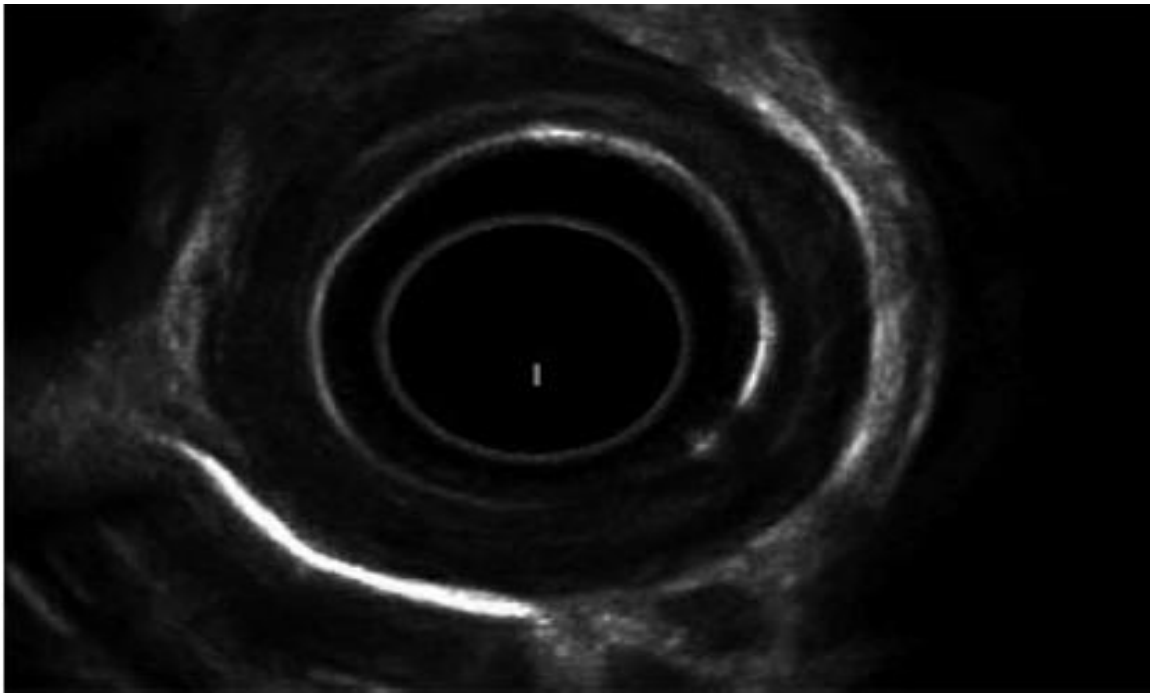


Figure 1.1 : EUS showing involvement of muscularis propria of esophagus

(iv) CT scan

Transmural damage and the extent of necrosis better delineated compared to early endoscopy[47].

A CT grading system has been proposed to predict esophageal stricture[48,49].

TABLE 1.3 : CT GRADING FOR CORROSIVE INJURY ESOPHAGUS.

Ryu et al[48]

Grade	Features
Grade 1	No definite swelling of esophageal wall
Grade 2	Edematous wall thickening without periesophageal soft tissue involvement
Grade 3	Edematous wall thickening + periesophageal soft tissue infiltration + well-demarcated tissue interface
Grade 4	Edematous wall thickening + periesophageal soft tissue infiltration + blurring of tissue interface or localized fluid collection around the esophagus or descending aorta

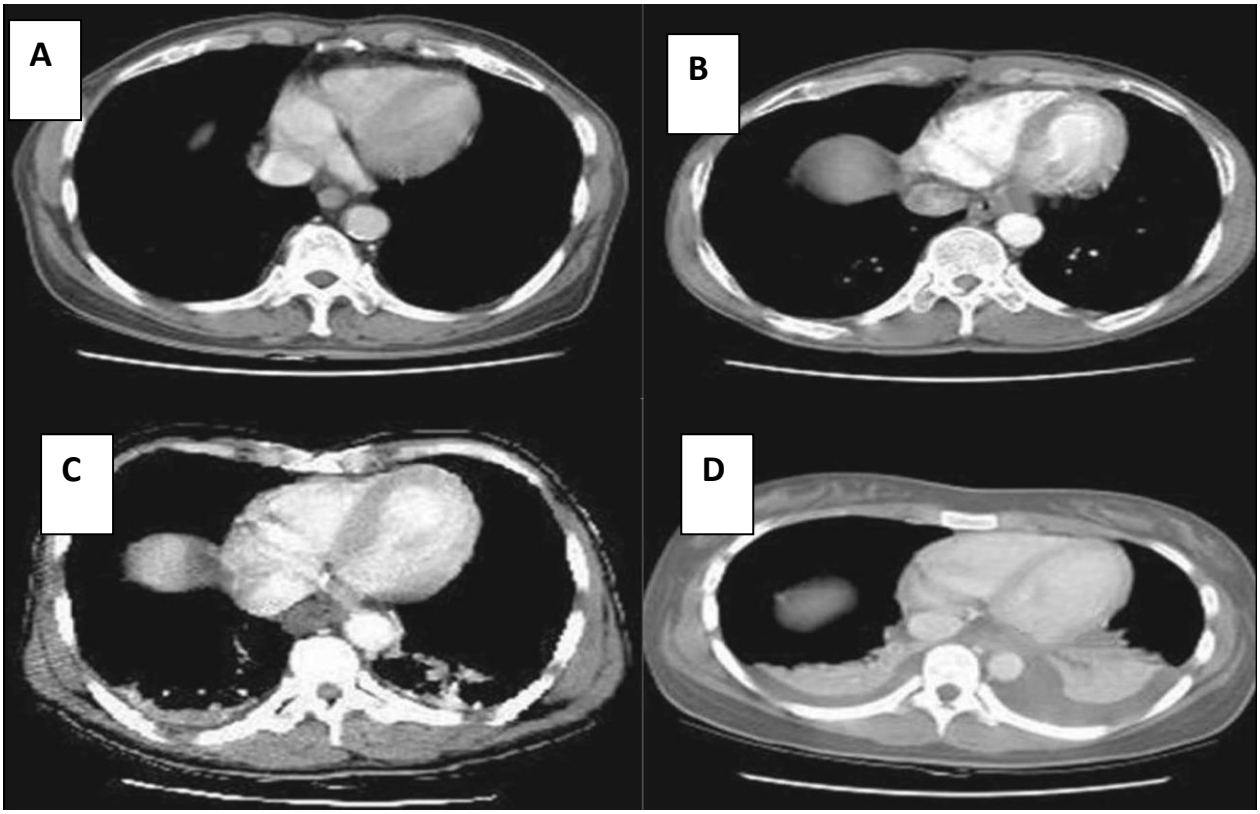


Figure 1.2:CT Chest showing A: Grade 1; B: Grade 2; C: Grade 3; D: Grade 4. Reproduced from Ryu *et al*[48].

Endoscopy

OGD is recommended in the first 12-48 h after caustic ingestion, though it is safe and reliable up to 96 h after the injury[50].

Endoscopy and even prophylactic dilatation though potentially hazardous have been performed 5 to 15 d after corrosive ingestion[51]. In patients who underwent prophylactic early bougienage, strictures had resolved after 6 months of dilatation, whereas in those in whom dilation began after stricture development, stricture resolution did not occur for > a year[51].

Though early dilatations do not abolish stricture formation, the strictures can resolve more easily. Endoscopy is contraindicated in patients with 3rd degree burns of hypopharynx , suspicion of perforation, supraglottic or epiglottic burns with edema & stridor.

Endoscopic classification is important for prognosis and management. Zargar's classification[20] is widely used.

Table 1.4 : MODIFIED ZARGAR'S CLASSIFICATION OF CORROSIVE INJURY

GRADE	ENDOSCOPIC FINDINGS
0	Normal Mucosa
I	Oedema & hyperaemia of the mucosa
II A	Superficial ulceration, erosions, friability, blisters, exudates, hemorrhages, whitish membranes.
II B	Grade II A plus deep discrete or circumferential ulcerations
III A	Small scattered areas of multiple ulceration and areas of necrosis with brown-black or greyish discolouration.
III B	Extensive necrosis

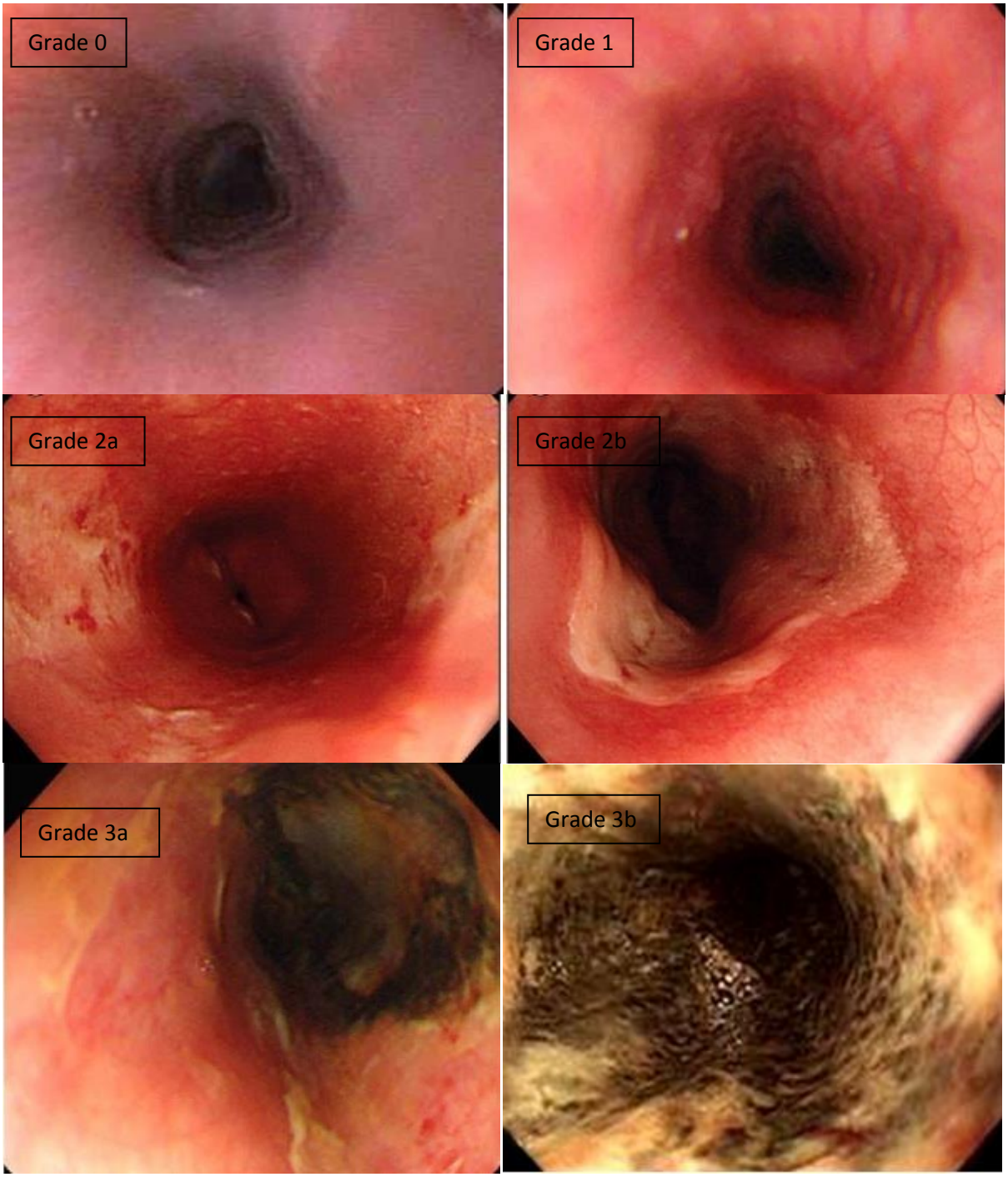


Figure 1.3 : Zargar's Endoscopic Grading Of Corrosive Injury Esophagus

Generally, patients with grade 0 and 1 injury do not develop delayed sequels. The degree of injury is an accurate predictor of systemic complications and death, with increase by each grade correlating with a 9-fold increase in mortality and morbidity[20].

MANAGEMENT

Acute management

Immediate treatment is usually conservative, as the severity of injury is determined within minutes after ingestion.

Hemodynamic stabilization and airway maintanence are top priorities.

Endotracheal intubation is indicated in patients with threatened airway. In patients with severe oropharangeal burns and glottis oedema, urgent ENT consultation should be obtained for tracheostomy. Induced emesis & Gastric lavage are contraindicated because of the risk of reexposure to the caustic agent and additional injury to the esophagus. The effectiveness of water and milk either as antidotes or to dilute the corrosive agents has never been proven. Weak acid or base, for pH neutralization is not recommended for fear of an exothermic reaction, which may increase the damage. Activated charcoal is contraindicated as it may obscure subsequent endoscopy.

Nasogastric tubes should never be placed blindly due to risk of perforation. They may be placed under fluoroscopic guidance and used as stent in severe circumferential burns, but their validity has never been proven.

The efficacy of proton-pump inhibitors and H2 blockers in minimizing oesophageal injury has not been proven, though an impressive healing after intravenous omeprazole infusion has been observed in a small prospective study[52].

The utility of corticosteroid in acute phase is controversial. Steroids are usually reserved for patients with symptoms involving the airway[53,54].

Broad-spectrum antibiotics are usually advised if corticosteroids are initiated, in lung involvement, in patients with complications like mediastinitis and perforation[55].

Patients with graded 1 and 2A are permitted oral intake and discharged within days. Patients with grade 2B or 3, should be admitted to intensive care unit and adequate nutritional support is required.

Early surgery

Immediate surgery is indicated in patients with perforation.

Some patients without perforation at admission may later develop necrosis, perforation and massive bleeding with devastating results.

Indications for emergency surgery rely more on clinical grounds than on radiological findings.

Disseminated intravascular coagulation, renal failure, acidosis and third degree esophageal burns are all indications for emergencysurgery[56,57].

The need to perform surgery for corrosive injuries has a long-term negative impact on survival and functional outcome. Emergency oesophageal resection per se, is an independent negative predictor of survival[58].

Late sequelae

Incidence of stricture following a grade 2B and 3 oesophageal burn can be as high as 71% and 100%, respectively. Strictures develop within 8 weeks in 80% of patients, but can occur as early as after 3 weeks or as late as after 1 year. Late sequelae of caustic gastric injury include intractable pain, gastric outlet obstruction, achlorhydria, mucosal metaplasia and carcinoma[59].

Stricture prevention

Steroids: Systemic administration of steroids is ineffective in preventing strictures. Intralesional triamcinolone injections can prevent strictures[60], but optimal dose, frequency, and best application techniques are still to be defined[61].

Nasogastric tube: Nasogastric tube placement may help to maintain patency of the esophageal lumen and enteral nutrition, however the tube can contribute to the development of long strictures and routine use is not recommended. NG tube may be a nidus for infection and may worsen GER in this patient population, with a consequent delay in mucosal healing.

Mitomycin C: Topical or injected Mitomycin C may be valuable in preventing strictures, but has deleterious adverse effects. Prospective studies are needed to determine the most effective concentration, duration and frequency of application[62].

Intraluminal stent: Silicone rubber[63]& polyflex stents[64] have been found useful in preventing stricture formation but the efficacy is < 50%, with a high migration rate (25%). Biodegradable stents made of poly-L-lactide or polydioxanone are under evaluation for benign strictures[65,66].

Stricture management

Endoscopic dilatation: Timely evaluation and dilatation play a central role in achieving a good outcome in stricture management[67]. Marked esophageal wall fibrosis and collagen deposition makes strictures complex and impedes effective dilation in late presentation.

Dilatation can be done with balloon or bougies. Savary bougies are more reliable than balloon dilators in fibrotic strictures such as old caustic stenosis or in long, tortuous strictures[68,69], and offer the advantage of feeling the dilatation by the operator.

The interval between dilatations varies from < 1 to 2-3 wk and usually 3-4 sessions are considered sufficient for durable results.

A cut-off value for unsuccessful dilatation treatment is difficult to define, especially in developing countries, where alternative surgical options are not widely available.

Risk of cancer

Risk of oesophageal adenocarcinoma and squamous cell carcinoma increases 1000-3000 times following caustic injury[70].The incidence ranges from 2% to 30%, with an latent period of 1 to 3 decades after ingestion.

Late surgery

Surgery for non-responding esophageal strictures:

When esophageal dilatation is not feasible or fails to provide an adequate esophageal caliber (15mm) in the long-term, esophageal replacement or

bypass should be considered. Removal of the native esophagus is advisable in children because of the risk of cancer in a long life period.

MATERIAL AND METHODS

This prospective study was conducted after institutional ethical clearance in the Department of Medical Gastroenterology, Rajiv Gandhi Government General Hospital, Chennai. The study period was from July 2013 to February 2014. Consecutive patients presenting with alleged history of corrosive ingestion were enrolled.

INCLUSION CRITERIA

1. History of corrosive ingestion less than 12hours before presentation to the hospital
2. Age >18 years

EXCLUSION CRITERIA

1. Late presentation (after >12hours ince ingestion)
2. Use of corticosteroids
3. Normal endoscopic study during initial OGD
4. Corrosive injury esophagus Grade 3b
5. Patients who are unstable for OGD

DATA COLLECTION

Data was collected prospectively on patient's demographics, type of corrosive agent, amount of corrosive ingestion, clinical presentation, & duration of hospitalisation.

Initial OGD was performed in stable patients within 24 hours of consumption of corrosive agent.

Grading of corrosive injury was done using Modified Zargar Classification.

NG tube was placed under fluoroscopic guidance in patients with grade 2b and 3a injury.

All patients with mucosal injury received 80mgs of pantoprazole stat followed by 8mg/hr infusion for 72 hours. All patients in the study were kept nil by mouth till second endoscopy.

Second endoscopy which was limited to screening of esophagus was performed by the same endoscopist, 72 hours after pantoprazole infusion in order to assess the mucosal healing compared to first endoscopy.

During the study period, 107 patients presenting with alleged history of corrosive injury were screened.

Of these 52 patients were excluded due to the following reasons – initial endoscopy revealed normal study (42), unwilling for second endoscopy (4), unstable for the procedure- Stridor(2) Endotracheal intubation with hemodynamic instability (1), initial endoscopy showed grade IIIB injury (3).

Hence, totally 55 patients were enrolled for the study.

The demographic profile of the patients included in the study is as follows

Table2.1: Demographic Profile			
Age Distribution	Age in years	Number of cases	Percentage
	≤ 20	07	12.7%
	21- 30	28	50.9%
	31-40	10	18.1%
	41-50	05	9.0%
	51-60	03	5.4%
	61-70	02	3.6%

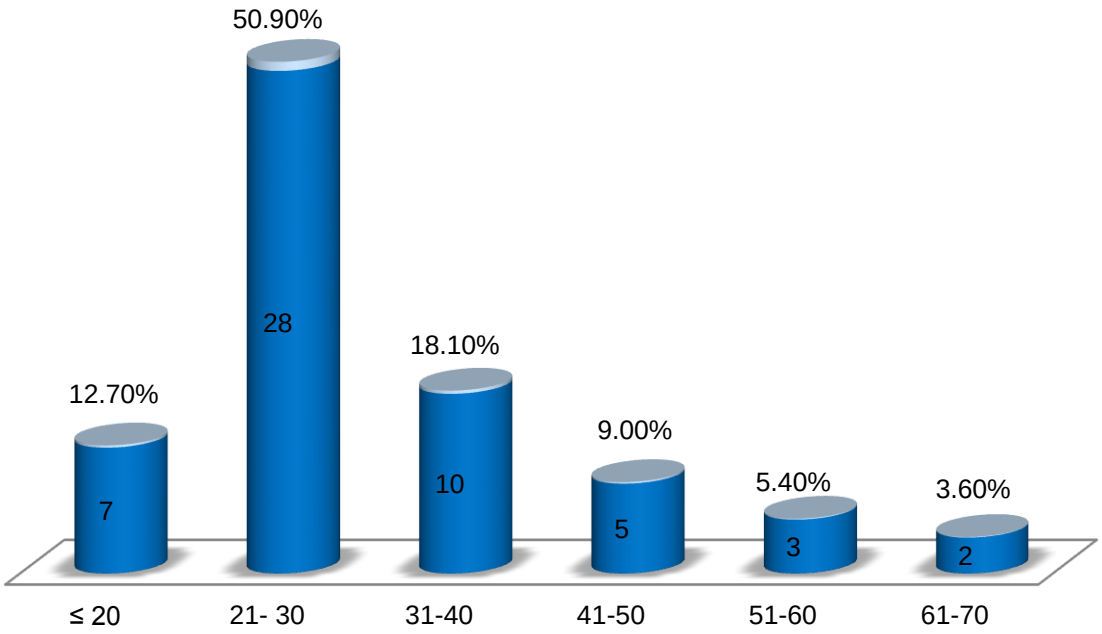


Figure2.1: Graph showing Age Distribution

Table 2.2 : Demographic profile			
Sex Distribution	Sex	Number of Cases	Percentage
	Male	30	54.5%
	Female	25	45.4%

Figure2.2: Piechart showing Sex Distribution

Table 2.3 and 2.4 showing the type of agent and intent of consumption respectively.

Table 2.3 : Agent Consumed		
	Number	Percentage
Acid	18	32.7
Alkali	37	67.2

Table 2.4: Intent of corrosive ingestion		
Sex	Accidental(n)	Suicidal(n)
Male	03 (10%)	27 (90%)
Female	01 (4%)	24 (96%)

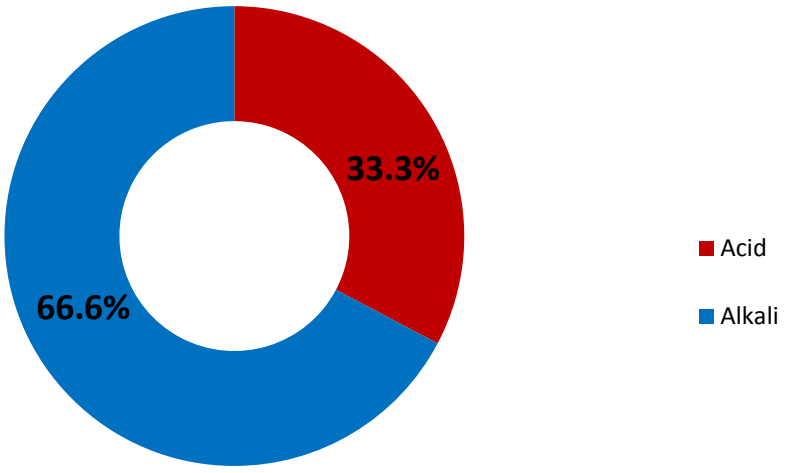


Figure 2.3: Chart showing agent Consumed

Table 2.5 showing the approximate amount of caustic agent consumed.

Table2.5: Amount of Corrosive agent consumed		
Approximate amount	Number	Percentage
<15ml	20	37
15-30ml	20	37
30-50ml	10	18.5
>50ml	5	9.0

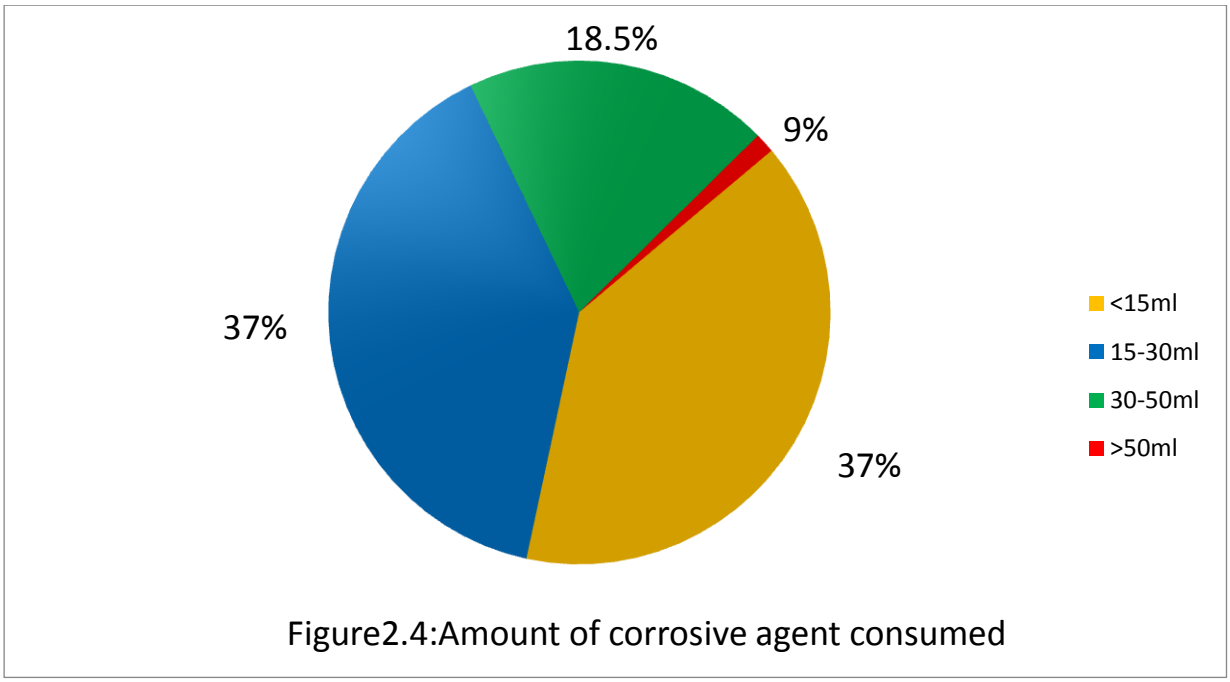


Figure2.4:Amount of corrosive agent consumed

Table 2.6 showing clinical presentation in all 55 patients

Table2.6 : Clinical Presentation		
	Number	Percentage
Oropharangeal ulcerations	8	14.5
Hypersalivation	38	69.0
Vomiting	55	100
Hemetemesis	20	36.3
Melena	4	7.2
Odynophagia	40	72.7
Retrosternal Chest pain	26	47.2
Epigastric Pain	38	69.0

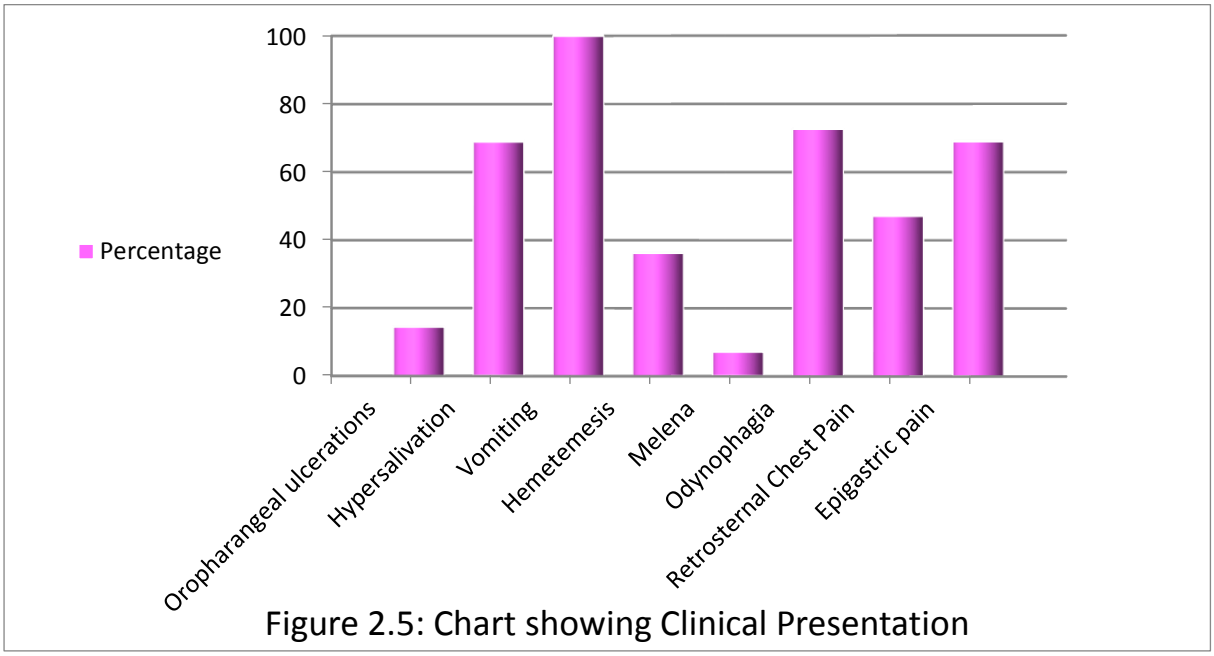


Figure 2.5: Chart showing Clinical Presentation

Table 2.7: Showing clinical presentation according to agent consumed				
	Acid		**Alkali**	
	Number	**Percentage**	**Number**	**Percentage**
Oropharangeal ulcerations	6	33.3%	2	5.4%
Hypersalivation	14	77.7%	24	64.8%
Vomiting	18	100%	37	100%
Hemetemesis	6	33.3%	14	37.8%
Melena	1	5.5%	3	8.1%
Odynophagia	16	88.8%	24	64.8%
Retrosternal Chest pain	13	72.2%	13	35.1%
Epigastric Pain	13	72.2%	25	67.5%

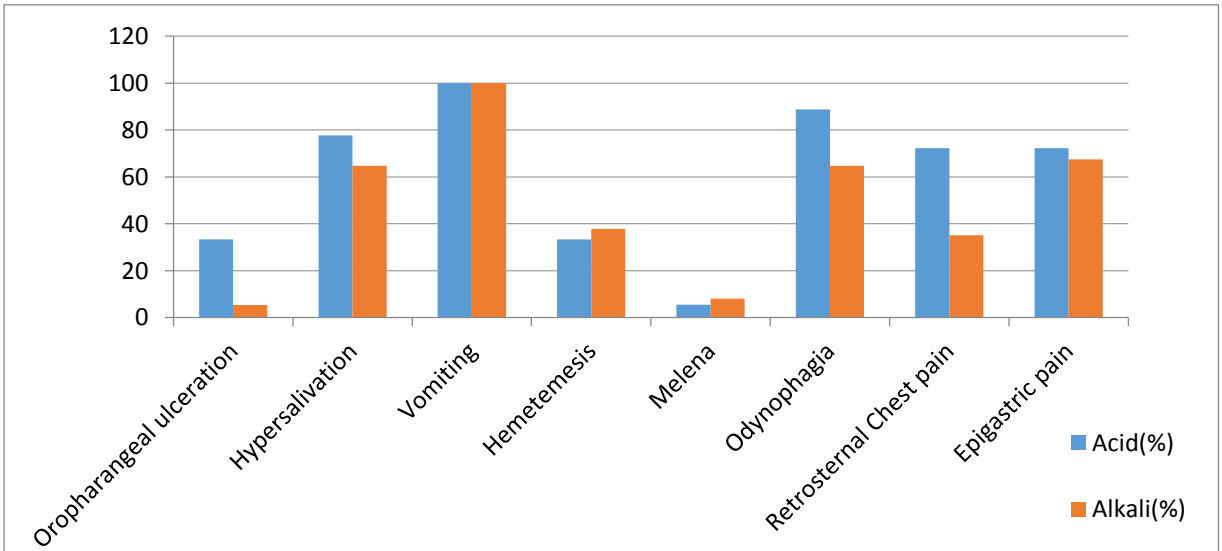


Figure 2.6:Chart showing clinical presentation according to agent consumed

Of the 20 patients who had presented with hematemesis, 16 patients had minor upper GI bleed and did not require blood transfusion,

4 patients had moderate upper GI bleed requiring blood transfusion.

6 patients complained of shortness of breath, however were not tachypnoeic and did not have stridor, dysphonia or aphonia, respiratory system and cardiovascular system examination were normal, chest X ray and Arterial blood gas analysis were within normal limits.

RESULTS

Table3.1: Injury Grade during initial OGD		
Zargar's Grade	Number	Percentage
Grade 1	24	43.6%
Grade 2a	16	29.0%
Grade 2b	09	16.3%
Grade 3a	06	10.9%

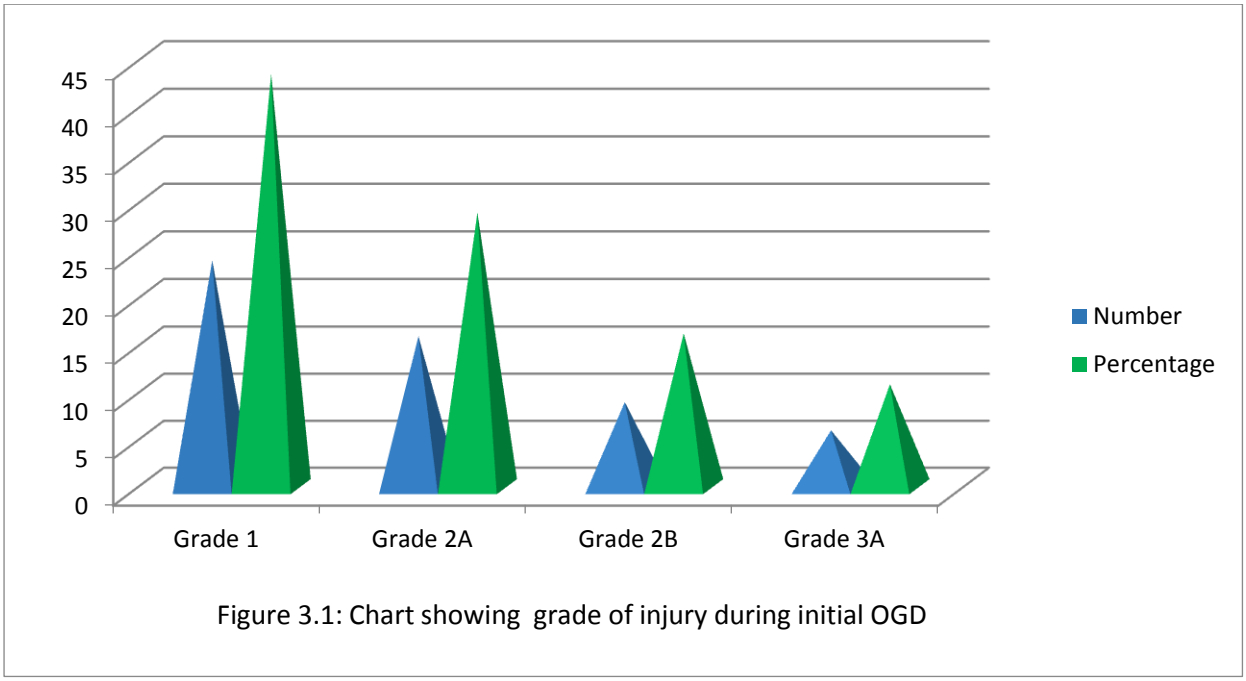


Figure 3.1: Chart showing grade of injury during initial OGD

Table3.2: Injury Grade after high dose pantoprazole		
Zargar's Grade	Number	Percentage
Grade 0	31	56.4
Grade 1	14	25.5
Grade 2a	4	7.2
Grade 2b	4	7.2
Grade 3a	2	3.6
Grade 3b	0	0

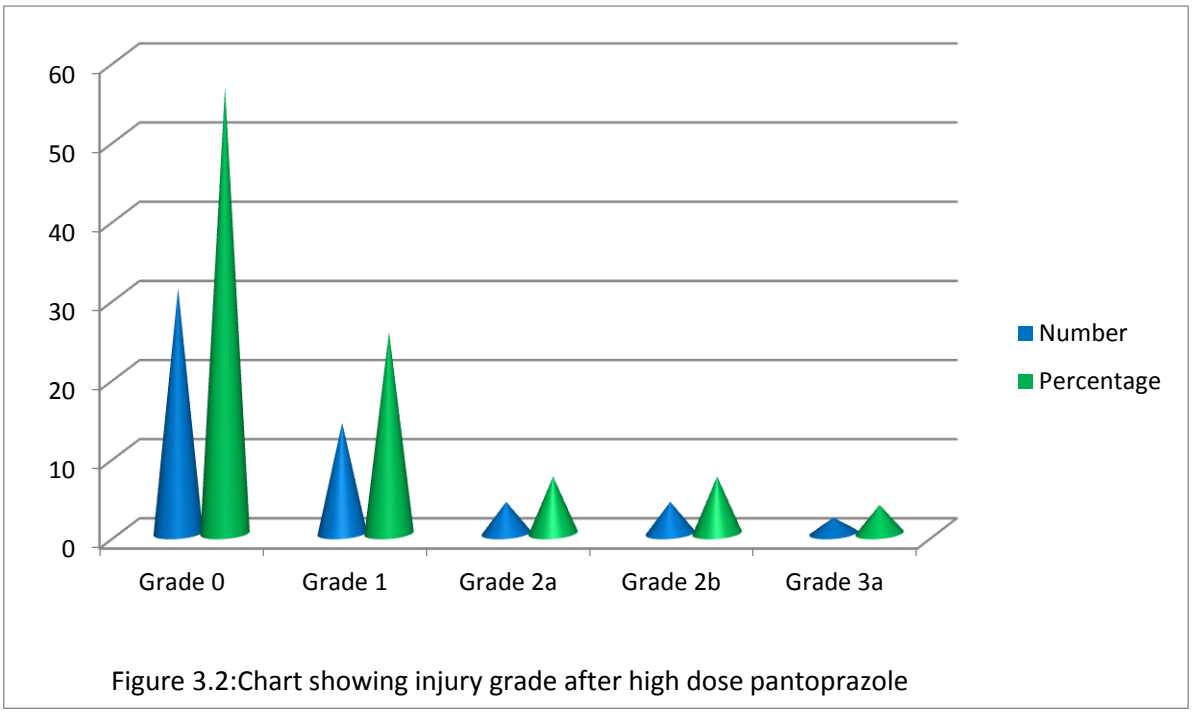


Figure 3.2:Chart showing injury grade after high dose pantoprazole

Table3.3 : Outcome of Grade 1 injury after pantoprazole					
Before			After		
Grade	Number	Percentage	Grade	Number	Percentage
1	24	43.46	0	24	100
			1	0	0
			2a-3b	0	0

Table3.4 : Outcome of grade 2a injury after pantoprazole					
Before			After		
Grade	Number	Percentage	Grade	Number	Percentage
2a	16	29.0	0	7	43.75
			1	9	56.25
			2a-3b	0	0

Table3.5 : Outcome of Grade 2b injury after pantoprazole					
Before			After		
Grade	Number	Percentage	Grade	Number	Percentage
2b	9	16.3	0	0	0
			1	5	55.5
			2a	4	44.4
			2b-3b	0	0

Table3.6 : Outcome of Grade 3a injury after pantoprazole					
Before			After		
Grade	Number	Percentage	Grade	Number	Percentage
3a	6	10.9	0-2a	0	0
			2b	4	66.6
			3a	2	33.3
			3b	0	0

Figure 3.3 showing outcome in terms of mucosal healing after high dose pantoprazole across all grades

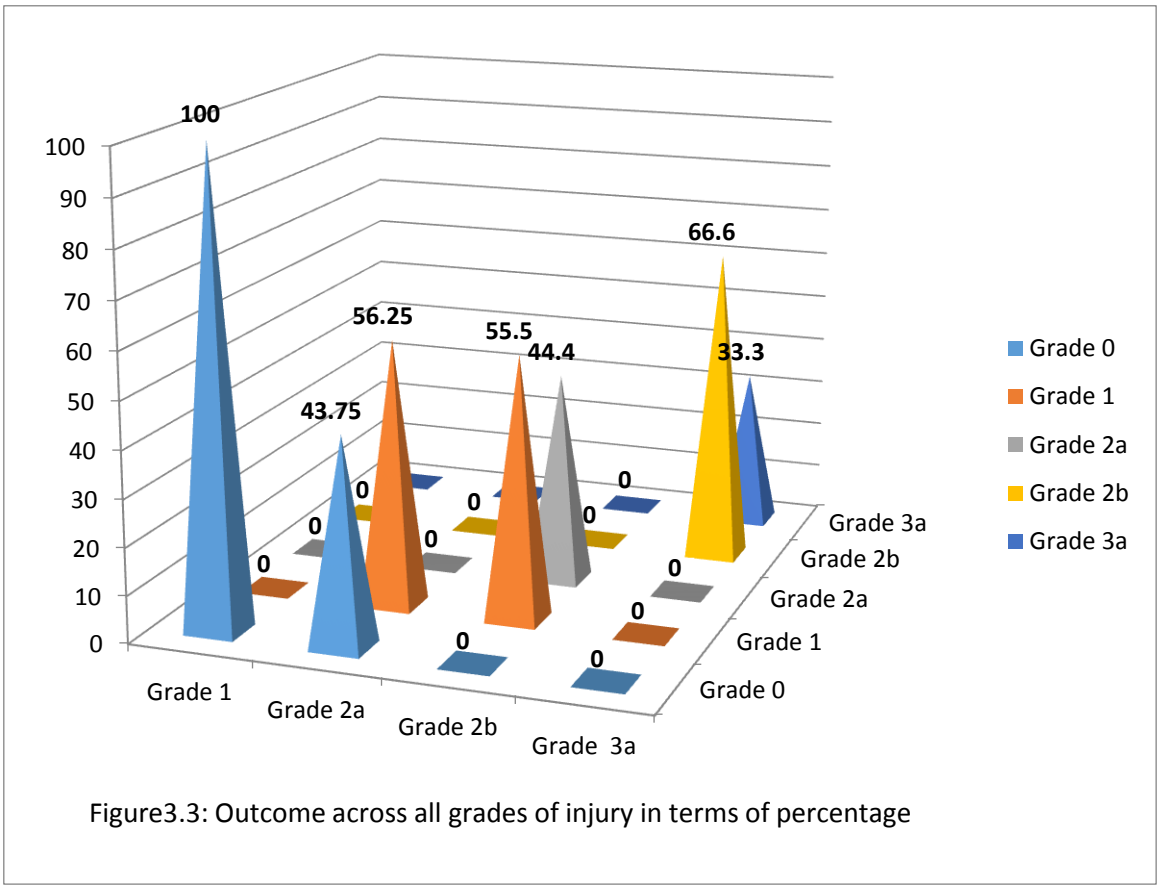


Figure3.3: Outcome across all grades of injury in terms of percentage

Figure 3.4 & 3.5 showing Box plot graph comparison of grade of injury before and after pantoprazole.

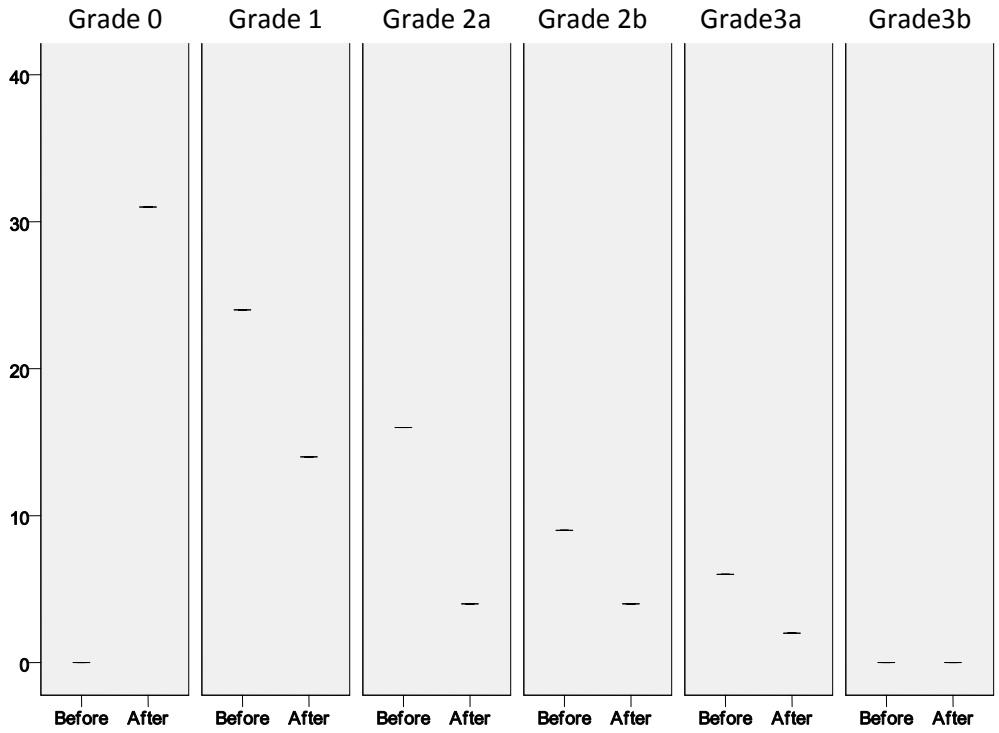


Figure3.4: Comparison of injury grade before and after pantoprazole (in terms of number, n55)

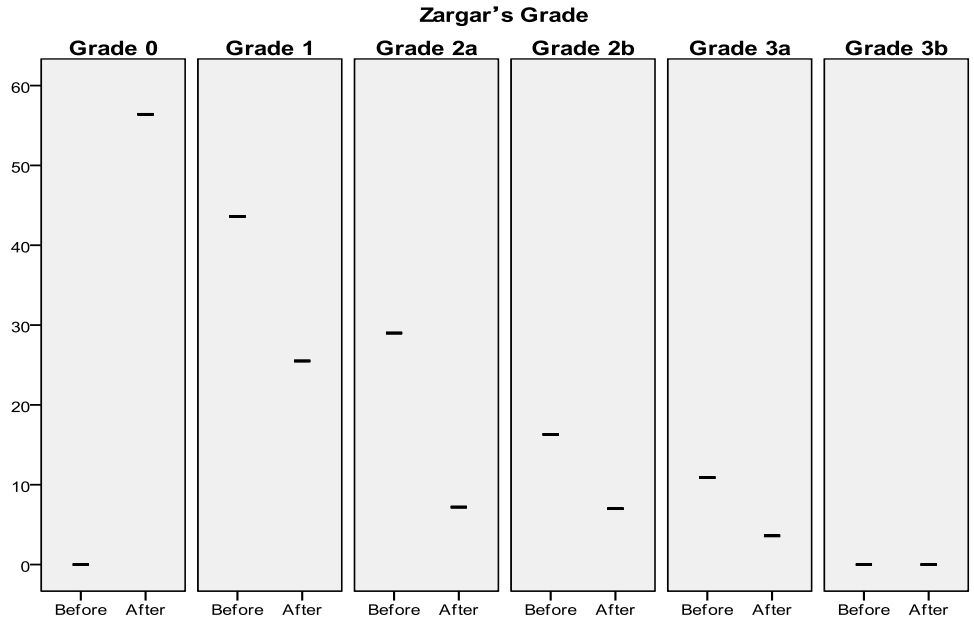


Figure 3.5: Comparison of injury grade before and after pantoprazole (in terms of percentage)

Figure 3.4 & 3.5 shows the boxplot summaries of the individual variable before and after high dose pantoprazole in patients with corrosive injury of oesophagus. Grade 0 and 3B being the exclusion criteria, there are no patients with this grade before enrolment.

A visual inspection of the figure shows tremendous improvement in the Zargar Grading “after” high dose pantoprazole when compared to the grade at initial endoscopy. There was a overall phenomenal improvement of 56.4 %(N=31) of patients to Grade 0 after the administration of pantoprazole. Mucosal healing was noted across all grades, though with varying frequency.

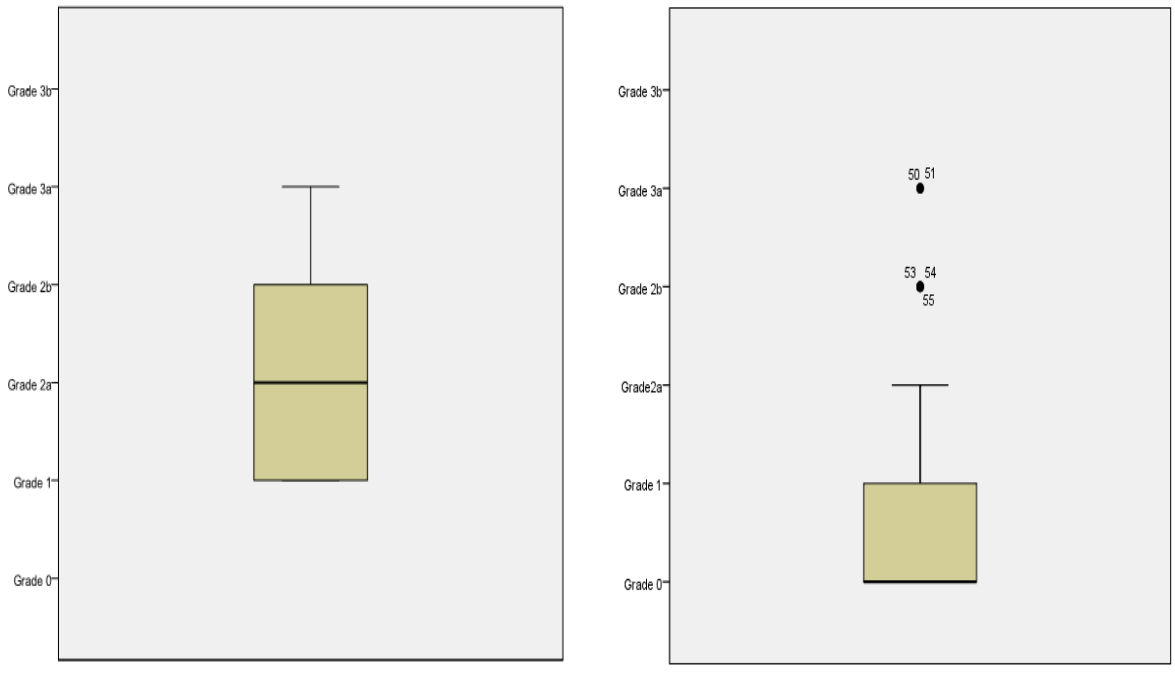


Figure 3.6: Boxplot showing endoscopic grade of 55 patients before and after high dose pantoprazole

Paired Comparison t - test

t- test result shows significant improvement in the mucosal healing after high dose of pantoprazole in acute corrosive injury of esophagus. For statistical analysis of mean, each grade of injury for assigned a (variable) number in ascending order viz Grade 0=1,Grade 1=2,Grade 2a=3,Grade 2b=4,Grade 3a=5,Grade 3b=6 .

The analysis of the mean values in terms of the Zargar grading before pantoprazole was 2.95 with a standard deviation of 1.026 (*M* = 2.95, *SD* = 1.026),

The mean values after pantoprazole was 1.76 with a standard deviation of 1.10 (*M* = 1.76, *SD* = 1.10). The level of significance *t*(54) = 18.453, *p* < .001.

Table 3.7: showing comparison of mean mucosal healing before and after pantoprazole

	Mean	N	Std. Deviation	t	df	Sig. (2-tailed)
Before pantoprazole	2.95	55	1.026	18.453	54	0.000
After pantoprazole	1.76	55	1.105			

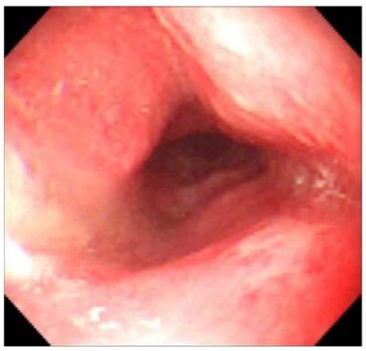
IMAGES

Before Pantoprazole –

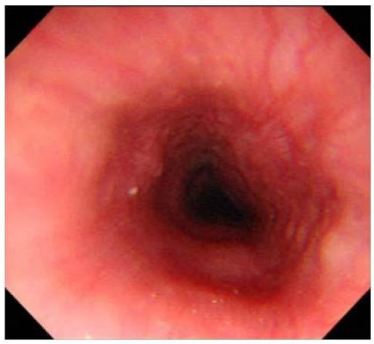
After Pantoprazole-

Patient :16

Grade 2a

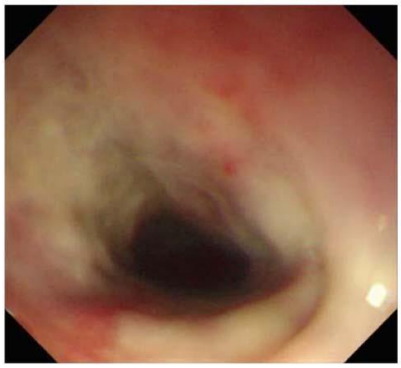


Grade 0



Patient: 4

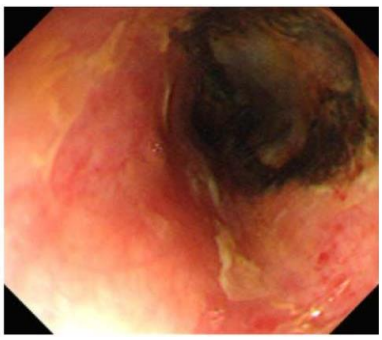
Grade 2b



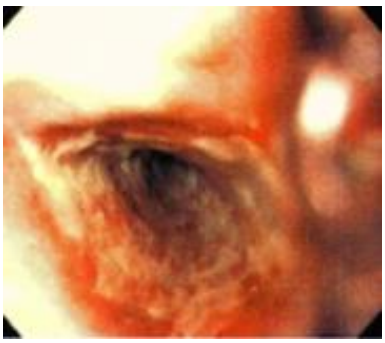
Grade 2a

Patient : 8

Grade 3a



Grade 2b



DISCUSSION

This is the first Indian study that has attempted to assess the efficacy of intravenous high dose pantoprazole in causing mucosal healing in acute corrosive injury of esophagus.

The only other prospective study to date in humans was from turkey by B.Cakal et al[52]. They have showed impressive mucosal healing in patients with acute corrosive injury of esophagus following high dose omeprazole, however their sample size was very small viz 13 patients.

Our study was restricted to the adult population, 50.9% of the patients in the study were between 21-30 years, which is the most productive phase of life.

Similar to our study, the mean age was 26 & 26.5 in the study from India by Zargar et al[20] and Gupta et al[22].

Severe corrosive injury is associated with lifelong complications, poor quality of life, adverse impact on social and economic life.

Overall > 90% of ingestion in our study was suicidal, which is much higher than that observed by Zargar et al[20]. Widespread and easy availability of corrosives is likely responsible for increasing incidence of suicidal corrosive ingestion.

Study from Puducherry[23] and Chandigarh[22] showed acid ingestion to be more common (82.6 & 83.4 respectively) than alkali. However our study showed Alkali poisoning (66.6%) to be more common than acid poisoning (33.3%). Most common agents were common household agents like toilet cleaners, dish wash agents, fabric stain removers, disinfectant surface cleaners and detergents.

All patients in our study population had spontaneous or induced vomiting immediately following ingestion. ~70% of the patients had epigastric pain, odynophagia and excessive salivation. 36% of the patients developed hemetemesis, however only 7% of the patients required blood transfusion. All 4 patients who needed blood transfusion had grade 3a injury on initial endoscopy. Of the 4 patients 2 showed improvement to grade 2b following high dose pantoprazole.

Only 10.9% of the patients had esophageal necrosis during the initial OGD. 2/3rd of the patients in the study had minor injuries viz Grade 1 and 2a. In our study the severity of injury was almost equally distributed irrespective of the agent consumed.

18 patients in the study had acid ingestion, of which 2(11%) had grade 3a, 3(16.6%) patients had grade 2b, 4(22%) patients had grade 2a and 9(50%) patients had grade 1 esophageal injury.

37 patients had alkali ingestion, of which 4(10.8%) had grade 3a,

6 (16.2%) patients had grade 2b, 12 (32%) had grade 2a and 15(40%) of the patients had grade 1 oesophageal injury.

Cheng et al[71] in their series of 273 patients reported that grade 3 injuries are more common, accounting for 44% of the cases in their study. This is in contrast to our study, in which grade 1 injury was more common accounting for 43% of cases. This may be attributable to the type and concentration corrosive agent that are easily available in different geographical regions.

Following high dose pantoprazole, endoscopic mucosal healing was observed across all groups by 1-2 grades. All 24 patients who had grade 1 injury showed normal endoscopic appearance after 72 hours of high dose pantoprazole. Among patients who had grade 2a injury at initial endoscopy, 43.7% improved by 2 grades to grade 0 & 56.2% improved by one grade to grade 1. Among patient who had grade 2b injury,55.5% improved by two grades and 44.4% improved by one grade. 2 patients with grade 3a injury did not show any improvement after 72 hours of high dose pantoprazole, however 4 patients(66.6%) showed improvement by one grade to grade 2b.

The mean injury grade improved from 2.95 to 1.76 after high dose pantopazole (M=1.76,SD=1.10) with a p value of 0.000

Our study has shown that use of high dose pantoprazole for 72 hours in acute corrosive injury of oesophagus can facilitate mucosal healing by 1-2 grades. The healing was more impressive with minor grades.

Cakal et al[52] showed impressive mucosal healing in patients with grade 1 and 2a, where all patients all normal endoscopy findings after 72 hours of omeprazole infusion. They also showed improvement by 2 grades in patients with grade 2b. One patient with grade 3a showed improvement by 3 grades to grade 1 at second endoscopy, however in another patient with grade 3a injury there was no improvement at second endoscopy.

The above findings are comparable to our study and suggests that high dose pantoprazole are definitely effective in treatment of patients with minor injuries. However only a subgroup of patients with advanced grade showed improvement after high dose proton pump inhibitors.

The exact mechanism by which proton pump inhibitors cause mucosal healing in corrosive injury is not known.

Proton pump inhibitors, by decreasing acid secretion in stomach may limit reflux induced worsening of injury.

Proton pump inhibitors has been shown to accelerate the microvascular and connective tissue regeneration through an increase in the concentration of fibroblast growth factor, myofibroblasts[72,73].

In addition, Omeprazole has been shown to increase hydroxyproline concentration in esophageal mucosa which in turn provides protection from esophageal burns[74].

Furthermore, one experimental study in rats has shown that omeprazole may prevent inflammation in the early phase of corrosive burn following the ingestion of acid and/or alkali[8].

The above mentioned pleotropic effects of proton pump inhibitors probably plays a role in causing mucosal healing in acute corrosive injury of esophagus.

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CONCLUSION

Treatment of corrosive injury is controversial and there are no definitive protocols.

Intravenous proton pump inhibitors are widely used in treatment of corrosive injuries despite lack of evidence.

Our study has demonstrated that effective mucosal healing can be achieved by intravenous proton pump inhibitor infusion.

Though majority of patients in our study had minor grade of injury, 27% of the patients included in the study had 2b and 3a injury.

When analysis is confined to these patients, 53% showed improvement by 1 grade, 33.3% showed improvement by 2 grades and 13.3% did not show improvement.

Therefore it is evident from above that high dose pantoprazole is also beneficial in patients with higher grade of injury and may prevent late complications in these patients.

A larger randomised placebo controlled trial in patients with higher grade of oesophageal injury is needed to determine if high dose pantoprazole should form standard of care in treatment of patients with acute corrosive injury of oesophagus.

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INFORMED CONSENT

DESCRIPTION: You are invited to participate in a research study on caustic injury esophagus. We are going to assess healing of caustic injury.

PROCEDURES: You will be asked to undergo upper GI endoscopy on two occasions. It requires fasting for atleast 6 hours,procedure will not require any sedation and will take maximum 10 minutes for completion.

RISKS AND BENEFITS: Risks include aspiration, esophageal perforation which occur rarely It will help in prognostication , help in predicting need for surgery in the future.

TIME INVOLVEMENT: Your participation in this study will be same as the time taken for management of caustic injury otherwise.

PAYMENTS: You will not be paid to participate in this study.

I, _____ , hereby consent to undergo upper GI endoscopy for the purpose of estimating extent and assessing healing of esophageal injury. I understand the risks and benefit of the procedure. I have understood that the findings will be used for research and the results will not be available to me.

Signature of the patient

Name of the Patient

Witness – Signature, Name and address

MUCOSAL HEALING AFTER HIGH DOSE PPI IN ACUTE CORROSIVE INJURY ESOPHAGUS

PROFORMA FOR DATA COLLECTION

S/No	DOA	IP NO.	TOXI NO.	DOD

DEMOGRAPHY	
NAME	
AGE/SEX	
ADDRESS	
CONTACT NO.	

AGENT CONSUMED	CONSTITUENT	AMOUNT	INTENT

CLINICAL FEATURES	
AT PRESENTATION	AFTER 72-96 HRS
Oral pharyngeal burns	
Hypersalivation	
Odynophagia	
Vomiting immediately after consumption	
Hemetemesis	
Melena	
Retrosternal Chest pain	
Pain Abdomen	
Miscellaneous	

INVESTIGATIONS	
CBC (HB/TC/DC/ESR/Plt Count)	
RFT	
LFT	
CxR	
AxR	

ENDOSCOPIC FINDINGS	
FIRST OGD	
SECOND ESOPHAGOSCOPY	

MANAGEMENT	
HIGH DOSE PPI	
STANDARD DOSE PPI	
IV ANTIBIOTICS	
IV CORTICOSTEROIDS	
SUCRALFATE	
NGT AFTER INITIAL OGD	

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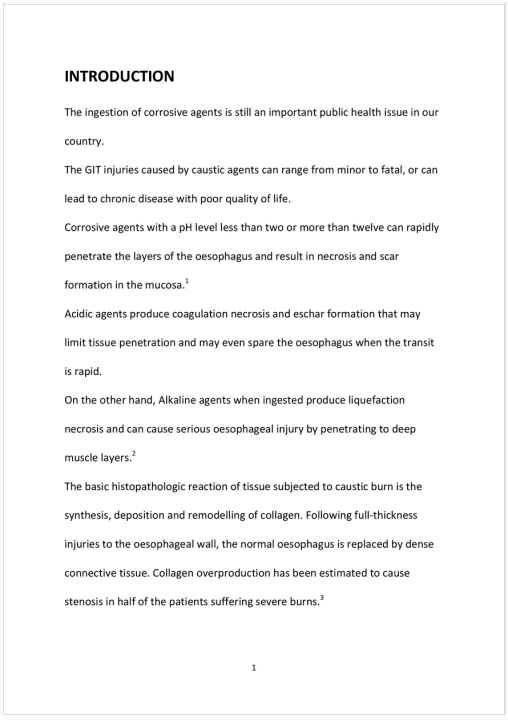

INTRODUCTION

The ingestion of corrosive agents is still an important public health issue in our country.

The GIT injuries caused by caustic agents can range from minor to fatal, or can lead to chronic disease with poor quality of life.

Corrosive agents with a pH level less than two or more than twelve can rapidly penetrate the layers of the oesophagus and result in necrosis and scar formation in the mucosa.[1]

Acidic agents produce coagulation necrosis and eschar formation that may limit tissue penetration and may even spare the oesophagus when the transit is rapid.

On the other hand, Alkaline agents when ingested produce liquefaction necrosis and can cause serious oesophageal injury by penetrating to deep muscle layers.[2]

The basic histopathologic reaction of tissue subjected to caustic burn is the synthesis, deposition and remodelling of collagen. Following full-thickness injuries to the oesophageal wall, the normal oesophagus is replaced by dense connective tissue. Collagen overproduction has been estimated to cause stenosis in half of the patients suffering severe burns.[3]

1

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Acidic agents produce coagulation necrosis and eschar formation that may

SR NO	S/No.	NAME	AGE	SEX	INTENT	AGENT	AMOUNT	OROPH. ULCERATION
1	1	ANAND	25	M	SUICIDAL	ALKALI	< 15 ml	
2	2	BHUVA	22	F	SUICIDAL	ALKALI	30 - 50 ml	
3	3	BOOPTHY	32	M	SUICIDAL	ACID	15 - 30 ml	
4	4	CAVERI	24	F	SUICIDAL	ACID	15 - 30 ml	*
5	5	DEEPAN	23	M	SUICIDAL	ACID	30 - 50 ml	*
6	6	DEVI	24	F	SUICIDAL	ALKALI	15 - 30 ml	
7	7	DHEEPA	20	F	SUICIDAL	ALKALI	15 - 30 ml	
8	8	DURAI	39	M	SUICIDAL	ACID	15 - 30 ml	*
9	9	EDWIN	38	M	SUICIDAL	ALKALI	15 - 30 ml	
10	10	GAJEN	67	M	SUICIDAL	ACID	< 15 ml	*
11	11	GOPAL	50	M	SUICIDAL	ALKALI	15 - 30 ml	
12	12	HARIK	56	M	ACCIDENT	ACID	< 15 ml	
13	13	HARINI	20	F	SUICIDAL	ALKALI	15 - 30 ml	
14	14	ILKYA	23	F	SUICIDAL	ACID	< 15 ml	
15	15	JIVITA	21	F	SUICIDAL	ALKALI	< 15 ml	
16	16	KANNG	27	F	SUICIDAL	ALKALI	15 - 30 ml	
17	17	KARTHIK	26	M	SUICIDAL	ALKALI	30 - 50 ml	
18	18	KARTHIK	53	M	SUICIDAL	ACID	< 15 ml	
19	19	KOTHNY	21	F	SUICIDAL	ALKALI	< 15 ml	
20	20	KUTTI	33	F	SUICIDAL	ALKALI	30 - 50 ml	
21	21	LAKSH	24	F	SUICIDAL	ALKALI	15 - 30 ml	
22	22	MAHE	40	F	SUICIDAL	ALKALI	15 - 30 ml	
23	23	MALAR	40	F	SUICIDAL	ACID	< 15 ml	
24	24	MARY	23	F	SUICIDAL	ALKALI	> 50 ml	
25	25	MOIDEEN	21	M	SUICIDAL	ALKALI	> 50 ml	*
26	26	NAZEER	25	M	SUICIDAL	ALKALI	> 50 ml	
27	27	NAZRIN	27	F	ACCIDENT	ALKALI	> 50 ml	
28	28	PARAM	49	M	ACCIDENT	ALKALI	30 - 50 ml	
29	29	PERUMAL	33	M	ACCIDENT	ALKALI	30 - 50 ml	
30	30	PREM	19	M	SUICIDAL	ALKALI	< 15 ml	
31	31	PUSHPA	35	F	SUICIDAL	ACID	< 15 ml	
32	32	RAJWR	21	F	SUICIDAL	ALKALI	15 - 30 ml	
33	33	RAM	57	M	SUICIDAL	ALKALI	< 15 ml	
34	34	RAMP	25	M	SUICIDAL	ACID	< 15 ml	
35	35	RANJITH	25	M	SUICIDAL	ALKALI	30 - 50 ml	
36	36	RAVI	22	M	SUICIDAL	ALKALI	15 - 30 ml	
37	37	RONALD	18	M	SUICIDAL	ALKALI	15 - 30 ml	
38	38	SANG	30	F	SUICIDAL	ACID	< 15 ml	
39	39	SARAN	46	M	SUICIDAL	ALKALI	30 - 50 ml	
40	40	SARATH	62	M	SUICIDAL	ACID	< 15 ml	
41	41	SHARM	18	F	SUICIDAL	ACID	< 15 ml	
42	42	SHIVA	32	M	SUICIDAL	ALKALI	15 - 30 ml	*
43	43	SHIVA	19	F	SUICIDAL	ACID	< 15 ml	*
44	44	SITA	30	F	SUICIDAL	ALKALI	< 15 ml	

45	45	SRINI	22	M	SUICIDAL	ACID	< 15 ml	*
46	46	SUDHA	26	F	SUICIDAL	ACID	< 15 ml	
47	47	THULSI	30	F	SUICIDAL	ALKALI	30 - 50 ml	
48	48	VAKU	22	F	SUICIDAL	ALKALI	15 - 30 ml	
49	49	VENKAT	19	M	SUICIDAL	ALKALI	> 50 ml	
50	50	VENU	28	M	SUICIDAL	ALKALI	30 - 50 ml	
51	51	VIGNESH	45	M	SUICIDAL	ALKALI	15 - 30 ml	
52	52	VIJYA	33	F	SUICIDAL	ACID	< 15 ml	
53	53	VINOD	42	M	SUICIDAL	ALKALI	30 - 50 ml	
54	54	VINOTH	23	M	SUICIDAL	ALKALI	15 - 30 ml	
55	55	VISHNU	30	M	SUICIDAL	ALKALI	15 - 30 ml	

CLINICAL PRESENTATION							GRADE DURING INITIAL O		
HYPER SALIVATION	VOMITING	HEMETEMESIS	MELENA	ODYNOPHAGIA	CHEST PAIN	EPIGASTRIC PAIN	1	2A	2B
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GD GRADE AFTER PANTAPRAZOLE

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